SIR,—Dr Peter V Scott and others (2 August, p 351) report favourably on the analgesic effect of subarachnoid morphine sulphate in 12 consecutive unselected mothers and suggest that the high rate of caesarean section (three cases) and forceps delivery (three cases) which followed was not associated with this method of treatment.

One important detail, however, was overlooked: the difference between primigravidae and multigravidae. Thus, all six operative deliveries were performed on seven primigravidae, whereas none was performed on five multigravidae included in the series. It cannot be reiterated too often that primigravidae and multigravidae behave almost as different biological species in labour, and nowhere is this more apparent than in the need for pain relief on the one hand and the likelihood of spontaneous delivery on the other. A parous woman needs little in the way of analgesia during labour, and is almost sure to deliver herself. The inclusion of multigravidae, therefore, has the effect of diluting the adverse effects of medication such as this. It would have been more realistic to highlight the fact that only one in seven primigravidae who received this form of treatment subsequently succeeded in delivering herself. There is no shortage of effective analgesic procedures—the question is are they safe? A sharp increase in the rate of operative intervention is a matter of the utmost concern to obstetricians and paediatricians alike.

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SIR,—The analgesia following the use of intrathecal morphine for obstetrics by Dr Peter V Scott and others (2 August, p 351) mirrors our results when intrathecal morphine is given for acute surgical pain. I would make one caveat. Dr Scott rightly emphasises the need for sterility of all equipment. In a series of over 50 patients who underwent various types of surgery we have found that preservative, bactericide, and antioxidant-free morphine is not a very stable product. Repeated autoclaving—for example, at the time of manufacture and then prior to use—may cause total loss of the analgesic potency of dilute (0.2 mg/ml) morphine. We now use morphine from ampoules that are double wrapped at the time of manufacture, and so are autoclaved only once.

Dr A A Spence's article on postoperative care, in the same issue of the *BMJ* (p 367), was of interest but the section devoted to postoperative pain is likely to become obsolete in the immediately foreseeable future. Intrathecal morphine abolishes all severe postoperative pain for a very prolonged period (24–48 h), so that the scandal of modern surgery to which he refers is unnecessary. Inter alia it also virtually eliminates the postoperative hypoxaemia to which Dr Spence also refers in his article. It is planned to publish full details of these observations in the near future.

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Sudden infant death syndrome and ventilatory control

SIR,—Your leading article (2 August, p 337) approaches the problem of sudden infant death

syndrome and its possible relationship to disordered ventilatory control in an inaccurate and misleading fashion.

In the first paragraph the statement is made that adverse social factors play a large part in the selection of victims. In the paper quoted,¹ however, no separation was made between sudden infant deaths where an adequate cause of death was found at necropsy from those where no explanation could be found (that is, sudden infant death syndrome). It is probable that adverse social factors act in the former group by a delay in the detection or realisation of the severity of a subsequently fatal illness. Without separating these two groups one cannot state that sudden infant death syndrome is more frequent in adverse social circumstances.

The relationship between disordered breathing patterns and so-called near-miss "cot death" has been investigated only after the event. We have already suggested from our own findings of prolonged apnoea in some cases of near-miss cot death² that it may be erroneous to assume that disordered breathing after a near-miss event was there prior to the episode of collapse. It is equally possible that some other disorder resulted in the collapsed state with subsequent damage to the respiratory control centres in the brain. Thus, although studies of infants who have suffered near-miss cot death episodes are of interest, they provide only circumstantial evidence. In the present state of knowledge, the statement in your leading article that it is "very likely that children with impaired chemoreceptor ventilatory drive have a greater than normal chance of succumbing to the sudden infant death syndrome" remains speculation. Apart from the finding of prolonged apnoea in one of 100 otherwise healthy preterm infants immediately prior to discharge from hospital,² there is no reported evidence that disordered ventilation occurs in otherwise healthy infants. Then to suggest that disordered ventilation is very likely to result in sudden infant death syndrome is presumptuous. It may cause parents to seek security from home apnoea alarm systems and subsequently suffer the serious side effects of such treatment,³ especially if disordered breathing is not a *primary* cause of sudden infant death syndrome.

Thus we agree that prospective studies of both ventilation and carbon dioxide responsiveness are required in large populations of otherwise healthy infants. A comparison of such measurements in infants who subsequently suffer sudden infant death syndrome and those who survive should answer the question of whether some healthy infants have abnormal and harmful ventilatory control mechanisms that place them at an excessive risk of sudden infant death syndrome. We are at present undertaking a prospective study of heart rate and breathing patterns in 15 000 babies using a 24-hour tape recording technique (supported by the British Heart Foundation).

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¹ Carpenter RG, Gardner A, McWeeny PM, Emery JL. *Arch Dis Child* 1977;52:606–12.

² Southall DP, Richards JM, Brown DJ, Johnston PGB, de Swiet M, Shinebourne EA. *Arch Dis Child* 1980;55:7–16.

³ Steinschneider A. *Pediatrics* 1976;58:1–4.

Febrile convulsions

SIR,—The NIH consensus statement on febrile convulsions is a very instructive account of the problem (26 July, p 277). I see about half a dozen cases a year in my general practice of 3400, which includes approximately 300 children under the age of 5.

The statement is made that initial investigations should include a complete paediatric and neurological examination; what does this mean? I do not send these children to hospital as a rule. I carry out a complete physical examination, which in my undergraduate days included a neurological examination. Should I be sending them all, immediately, to a paediatric unit for admission? If yes, then I am afraid that I shall have to ask the Health Service to build another paediatric hospital first, otherwise the deluge of cases from Lothian Region will swamp the existing facilities.

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Sudden cardiac death, cardioprotection, and beta-adrenoceptor-blocking agents

SIR,—At the present time enormous resource and energy are being directed towards the prevention of sudden cardiac death. The benefits to man if such endeavours are successful will be immense, as will be the financial implications of the long-term treatment that may be involved. For there to be maximum benefit to all parties it would seem of paramount importance, particularly at this stage, to maintain the strictest scientific, statistical, and commercial discipline.

The β -adrenergic blocking drugs particularly are being promoted for their "cardioprotective" properties. If "cardioprotection" is the effective control of arrhythmias, hypertension, and angina then the case is proved in the short and the long term. If however, cardioprotection refers to sudden cardiac death (as indeed it originally did) the case is not yet proved. Despite this, recent advertisements have made such claims, by implication, for at least two beta-blocking agents, neither of which have been used in published prevention studies.

More recently Dr N S Baber and Mr J A Lewis (5 July, p 59) have used the recommendation of Professor Geoffrey Rose (19 April, p 1088) concerning the inclusion of confidence limits to derive maximum benefit from apparently inconclusive results. They constructed a diagram summarising the efficacy of different beta-blocking agents in preventing sudden death. The data so treated were drawn from selected trials which differed not only in size but also in dose; inclusion criteria; the nature of end points; and above all the degree of attention given to proper controls, randomisation, and "blindness." Comparison of data in this way therefore is of limited value. Analysis and comparison of long-term clinical trials is described elsewhere.¹ It was particularly unfortunate that in the interest of saving space (editorial footnote, 2 August, p 387) some references were then omitted from the diagram. The result was that a non-randomised, inadequately controlled open study with a successful outcome for a particular drug was referenced (ref 2 in the letter by Dr Baber and Mr Lewis) whereas one with similar confidence limits but a less favourable outcome was not (second entry in figure). At this stage the display became at best unhelpful and at worst

provided temptation for use in promotional activities.

It is premature to assume that all beta-blocking drugs are equally effective in preventing sudden death and at present it is not known which subgroups of patients might benefit or for how long, and what doses are optimal. Every effort should be made to minimise speculation and over-simplification because of the danger of causing delays in the discovery of relevant mechanisms—or even worse of increasing scepticism and so depriving patients of effective treatment.

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¹ Peto R, Pike MC, et al. *Br J Cancer* 1977;35:1-39.

*.*The complete list of references for the diagram in the letter by Dr Baber and Mr Lewis is given below.—Ed, *BMJ*.

- a Sowton E. (Quoting Fucella LM). *Progr Cardiovasc Dis* 1968;10:561-74.
- b Norris RM, Caughey DE, Scott PJ. *Br Med J* 1968;iii:398-400.
- c Multicentre Trial. *Lancet* 1966;iii:1435.
- d Briant RB, Norris RM. *NZ Med J* 1970;71:135-8.
- e Baber NS, Evans DW, Howitt G, Thomas M, Wilson C. *Br Heart J* 1980;44:96-100.
- f Reynolds JL, Whitlock RML. *Br Heart J* 1972;34:252-9.
- g Balcon R, Jewitt DE, Davies JPH, Oram S. *Lancet* 1966;iii:917-20.
- h Clausen J, Felsby M, Schønau Jorgensen F, Lyager Nielsen B, Roin J, Strange B. *Lancet* 1966;iii:920-4.
- i Barber JM, Boyle DMCC, Chaturvedi NC, Singh N, Walsh MJ. *Acta Med Scand* 1975;suppl 587:213-9.
- j Barber JM, Murphy FM, Merrett JD. *Ulster Med J* 1967;36:127-30.
- k Mitchell JRA. *Br Heart J* 1978;40, suppl:88-93.
- l Sloman G, Stannard M. *Br Med J* 1967;iv:508-12.
- m Mitchell JRA. *Br Heart J* 1978;40, suppl:88-93.
- n Ahlmark G, Saetre H, Morsgren M. *Lancet* 1974;iii:1563.
- o Multicentre International Study. *Br Med J* 1975;iii:735-40.
- p Wilhelmsson C, Vedin JA, Wilhelmson L, Tibblin G, Werkö L. *Lancet* 1974;iii:1157-60.
- q Snow PJD. *Lancet* 1965;ii:551-3.
- r Kahler RL, Brill SJ, Perkins WE. In: Katus AA, Ross G, Hall YE, eds. *Cardiovascular Beta-adrenergic Responses*. Los Angeles: University of California Press, 1968:213.

the parallel increase in SOM scores with increasing consumption remained in both sexes even when these two questions were excluded and when only scores of the "respiratorily fit"² subjects were examined.

The tendency for scores to rise with increasing cigarette consumption points to a relationship between neuroticism and smoking, though which is cause and which is effect cannot be determined from these surveys alone.

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¹ Simpson FO, Nye ER, Bolli P, et al. *NZ Med J* 1978;87:379-82.

² Waal-Manning HJ, de Hamel FA. *NZ Med J* 1978;88:188-91.

Dietary prevention of ischaemic heart disease

SIR,—Evidence that dietary prevention of coronary heart disease has failed and recommendations that these measures should be abandoned have already been published.^{1,2} Professor Barry Lewis's efforts (19 July, p 177) to restore faith in such measures are either unconvincing or incorrect.

Earlier emphasis on "dangerous" cholesterol has now given way to HDL:LDL ratios. Comparative death rates over decades are very insecurely based as doctors progressively change their diagnostic habits. Neither Mackenzie nor Lewis was able to diagnose characteristic attacks of myocardial infarction in himself, lacking modern diagnostic confirmation. Improvement in the US mortality is part of a general increase in survival and coronary death rates have now fallen to the same level in the United States as in England and Wales,³ so we do not yet deserve a rebuke for our apparent inactivity.

The National Academy of Sciences and the National Research Council of America (surely the supreme court of science) have now reported⁴ that no prevention of coronary heart disease can be achieved by dietetic or drug reduction of blood cholesterol, except possibly when this is abnormal and there is a family history of such abnormality. The only response to this challenge by the polyunsaturated fat evangelists is not scientific but offensive innuendoes of malice and commercial influence, now rebutted by the National Academy of Sciences.

It is interesting to note the definition by Vergroesen⁵ (who was vice-chairman of the FAO-UN report committee): "Atherosclerosis . . . is characterised by deposits of esterified cholesterol . . . mostly from the blood lipoproteins . . . whose concentrations determine . . . the amount of accumulation subendothelially." Dr Vergroesen is director of research at Unilever and this definition provides the rationale of current commercial propaganda. The reasoning which makes this concept untenable can be summarised as follows:

(1) None of the best-conducted and statistically controlled trials of lipid-reducing polyunsaturated-fat diets or drugs have shown any preventive effects on the incidence or progress of coronary heart disease. Indeed, in the North Karelia (primary)⁶ and Sydney (secondary)⁷ prevention³ trials the controls fared better than groups treated with polyunsaturated fats.

(2) Only about 50% of atherosclerotic plaques contain any visible cholesterol, which is thus clearly not the cause. None of the spontaneously occurring hypercholesterolaemias (myxoedema, nephrosis, or even the rare homozygous form) are associated with any increased severity of ordinary coronary artery disease even after long exposure. The homozygous form can obstruct the coronary ostia only, leaving the coronary arteries virtually free of the disease.

(3) The butter- or cholesterol-fed rabbit lesion is part of a lipidosis in the arteries and other organs. The arterial lesions are really xanthomas and neither scar nor thrombosis, being wholly different in appearance and distribution from the human disease. It is only these dietary-induced lesions that regress with diet.

(4) Reproducing the familiar "jet lesion" and the localising effects of turbulence, experimental mechanical injury to the arterial lining produces at first a "pearly" fibrous plaque, which later contains cholesterol deposits at the naturally low levels of blood cholesterol of rabbits and sheep.^{8,9} Lipid accumulation is thus a secondary consequence, generated internally in the lesion, and it does not depend on flux of cholesterol from the plasma. It can ulcerate and thrombose and is the closest experimental model to the human disease. Coronary occlusive events in the individual patient are unrelated to blood cholesterol levels.

Thus atheroma almost certainly originates from platelet thrombosis and a cellular fibrous reaction on a site of endothelial injury and is quite unrelated to the cholesterol content of the blood.

It is deeply disturbing that some of our profession are stretching so much speculative and insecure evidence to support a dietetic theory no longer tenable by informed medical scientists. They wish to switch from our best dairy-based foods. They will try to change our agriculture.¹⁰ They have already persuaded the BBC to put out propaganda that these dietetic changes reduce coronary heart disease death rates in its recent home economics and nutritional programmes to schools. For this statement there is no evidence.

The threat is serious. As a profession we cannot any longer permit or connive at this deception of the public.

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¹ McMichael J. *Br Med J* 1979;ii:173.

² Ahrens EH. *Lancet* 1979;iii:1345.

³ Tunstall-Pedoe, H. *Lancet* 1980;ii:831.

⁴ National Academy of Sciences. *Towards healthful diets*. Washington: National Academy of Sciences, 1980.

⁵ Vergroesen AJ, Gottenbos JJ, Ten Hoor F. *Comprehensive Therapy* 1979;5 (12):19.

⁶ Salonen JT, Puska P, Mustaniemi H. *Br Med J* 1979;ii:1178-82.

⁷ Woodhill JM, Palmer AJ, Leelarthapin B, McGilchrist C, Blackett RB. In: Knitchowsky D, Paoletti R, Holmes W, eds. *Drugs, lipid metabolism and atherosclerosis*. New York: Plenum Press, 1979.

⁸ Moore S. *Lab Invest* 1973;29:478-87.

⁹ Stehbens WE. *Proc R Soc London B* 1974;185:357-73.

¹⁰ Centre for Agricultural Strategy, University of Reading. *National food policy in the UK*. University of Reading, 1979.

Psychoneurotic profiles of smokers and non-smokers

SIR,—A 1975 health survey in Milton, a small rural township in New Zealand,¹ gave very similar results² to those reported by Dr Andrew P Haines and his colleagues (14 June, p 1422) for the Northwick Park Heart Study (NPHS) participants.

The Middlesex Hospital Questionnaire subscale scores in the 992 New Zealand subjects (444 men and 548 women) grouped into smoking categories as in the study by Dr Haines and his colleagues were numerically similar to those of the NPHS. Subscale scores in a repeat survey in 1978 also were similar.

In our study² also, with a few exceptions, women scored higher on the subscales than did the men and heavy smokers scored higher than "never smokers," and there was a tendency for scores to rise with increasing cigarette consumption. This was especially so for SOM (somatic concomitants of anxiety), which we found to be the subscale which showed the stepwise score increase with increasing cigarette consumption most clearly and to the highest statistical significance ($p < 0.001$ analysis of variance).

Two of the questions asked in assessing SOM—namely, those concerning the occurrence of breathlessness and palpitations—could be associated with tobacco consumption in their own right. We found, however, that

SIR,—In his excellent review "Dietary prevention of ischaemic heart disease—a policy for the '80s" (19 July, p 177), Professor Barry Lewis refers to the strong positive correlations that have been established between plasma cholesterol concentrations and both mortality from ischaemic heart disease and the extent of coronary atherosclerosis. I deem it advisable to add a cautionary note, because the constituents measured as "cholesterol" could include other sterols.

Neither the older nor the newer clinical analytical methods in common use are specific for cholesterol: the enzyme cholesterol